RENAL STONES

Pages with reference to book, From 26 To 27

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Most common kidney stones are of calcium oxalate[1], 10 to 12% of them contain uric acid[2], while most have small amount of hydroxyappetite. Stones containing struvite, pure uric acid, combination of hydroxyappetite and calcium monohydroxy phosphate and a few containing cystine are also found. Calcium oxalate and calcium phosphate stones are black, grey or white, smaller than 1 cm in diameter, dense opaque and sharply circumscribed. Uric acid stones are white or orange and crystals are radio graphically transparent. Simple urinalysis can reveal the presence of crystals and provide clues to the stone type. To analyse the composition and differentiation of renal stones, polarization microscopy[3], infrared and X-ray diffraction[4] techniques are modern methods. CT scan is used to differentiate transparent stones from renal tissue or blood clots[5]. Renal stone formation depends upon the concentration of different salts in urine. In normal urine concentration of calcium oxalate salts is four times higher than its solubility, increased excretion of calcium and oxalate with less urinary volume increases calcium oxalate supersaturation and when it is 7-11 times the normal solubility, enucleation of calcium oxalate starts and possible surfaces for it in kidney are epithelial lining, cell debris, urinary cast and other crystals[6]. Any factor increasing heterogenous nuclei in tubular fluid or urine like epithelial injury lowers the upper metastable limit (super-saturation at which crystals first form), hyperuricosuria lowers the metastable limit by promoting calcium oxalate stone formation[7,8]. Hypercalciurea and hyperuricurea promote haematuria probably from crystal urea. Calcium phosphate super saturation occurs at urinary pH of 6.5 due to high proportion of divalent and trivalent phosphate ions. Microscopic nuclei can form stones by growing or aggregating into large clumps, but they cannot grow enough to anchor and occlude renal tubular lumen within 5 to 7 seconds, as they pass through nephron very rapidly but they can do so in one minute[9]. Kidney proteins inhibit all phases of crystalization. Nephrocalcin containing r-carboxyglutarnic acid, inhibit calcium oxalate stone formation[10-13]. Famm-Horsfall mucoprotein from renal thick ascending limb, inhibit aggregation of calciumoxalate crystals[14]. Uropontin by kidney inhibits the growth of calcium oxalate crystals. Patients with renal lithiasis present with intense pain radiating from flanks to anterior side of thigh -associated with nausea, vomiting[15], urinary frequency and dysuria as stone passes towards ureterovesical function. If it passes into bladder, decompression of urinary tract relieves pain spontaneously. Stone may cause obstructive uropathy if painless and remains undetected for a long time. Management of stones depends upon its site, size and type. Most ureteral stones less than 5mm in diameter pass readily, but 7 mm in diameter or larger stones have a poor chance of passing. Stones lodged in distal ureter, not progressing are best removed uretroscopically with a stone basket or disrupted in situ with extra corporeal shockwave lithotripsy (ESWL) [16-18]. Stones embedded in proximal ureter are pushed upwards in renal pelvis and disrupted by ESWL, it requires a cystoscopy to push the stone backwards by a catheter up the ureter. If double J stent is passed up the renal pelvis, it improves the chances of complete stone removal[18]. Percutane Our nephrolithotomy is needed if lithotripsy fails. Surgical ureterolithotomy should only be used as a last resort. Kidney stones that are less than 2 cm but more than 5 mm in diameter can best be treated with ESWL. Stones exceeding 2 cm or of 1 mm diameter lying in lower renal pole should be treated with percutaneous nephrolithotomy[17-19]. because of the use of lithotripsy alone leaves residual stones in 35 to 54% cases, whereas percutaneous nephrolithotomy succeeds in most[17-19]. Double J stent facilitates the complete drainage of fragments specially in case of large stones. Asymptomatic kidney stones of less than 5 mm should be left untreated. Etiological bases of

stones is another important aspect for its treatment and prevention of recurrence. People with primary hyperparathyroidism excrete a high fraction of dietary calcium and in these cases 1 a 25 dihydroxy colcalciferol, increases the intestinal absorption of calcium[20], raises renal tubular calcium resorption[21] and bone turnover leading to hypercalcaemia, hypercalciurea[22,23] and lowering of phosphorus resorption. Low serum phosphorus levels enhance the calciferol production, which in turn causes hypercalciurea[24,25]. In people with familial idiopathic hypercalciurea, erythrocyte calcium ATPase levels may vary directly with the level of urinary calcium excretion[26]. People having hypercalciurea as a result of high serum calcitriol levels caused by renal phosphate wasting is reversed by oral phosphate supplements. Pseudoxanthoma elasticum causes high levels of calcitriol and hypercalciurea[27], cystic fibrosis causes hypercalciurea and nephrocalcinosis[28]. Consanguineous marriages in hypercalciuric families raise the chances of hypercalciurea in successive generation with Increased rate of calcium excretion. Pak and colleagues[28] have proposed two types of idiopathic hypercalciurea. All patients with absorptive hypercalciurea have normal or low levels of serum parathyroid hormone and fasting urinarylevels below. 11 mg per dl. of creatinine clearance28. Thiazide diuretics lower urinary calcium excretion by increasing fractional calcium resorption through distal nephron and reducing intestinal absorption[29,30]. Renal tubular acidosis in patients with idiopathic hypercalciurea is due to papillary calcification, so mainly calcium phosphate stones are formed[31] and these people respond to thiazide diuretics whereas potassium alkali ratio are useful in patients with hypercalciurea due to metabolic acidosis[32]. Calcium oxalate stones are not very common but they can invade the urinary tract, cause pain, bleeding and then disperse, passed as gravel also called ‘crystalurea’. A simple dietary excess of oxalate from food like spinach, rhubarb, coco, beet, peanut, pepper, wheat germ and chocolates cause hyperoxalurea[33,34] (20-40 mg per day is normal). Malabsorption from small bowel from any cause including resection intrinsic disease, jejunal bypass, etc., expose the mucosa to detergents like bile salts and fatty acids which increase its permeability to oxalates[35,36]. Treatment includes reducing the dietary oxalate and fats[37], oral calcium supplements as carbonate salts and oral citrate supplements thatprecipitate oxalate in intestinal lumen. High fluid intake is also advised. Patients with hyperoxalurea respond to pyridoxin supplements and increased urinary volume to 3 litres. Uric acid stones are found in pure as well as combined with others. These are treated by raising the pH to 6-6.5 with potassium alkali salts. Allupurinol is beneficial in cases of 1200 mguric acid excretion per day. Urinary infection with bacteria that express urease cause struvite (magnesium ammonium phosphate) stone[38]. These are associated with bleeding obstruction or infection. They require removal; ESWL[39] or percutaneous nephrolithotomy to reduce their growth. Once patient is free of stone, antibiotics are beneficial. Increasing urinary pH and volume to 4 litre per day prevents cystine stones which is associated with cystineurea and dehydration. Penicillamine or tiopronin combined with cystine to form a soluble salt and reduce the stone formation. So they are also useful for its treatment[40].

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